Morris (R. T.)

INFECTIOUS APPENDICITIS.[1]

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THE factors in infectious appendicitis may be grouped as follows:

(1) Histological.

A thick layer of adenoid tissue.

(2) Anatomical.

(*a*) A soft, distensible tube of mucosa and adenoid tissue within a confining outer tube of muscle and peritonæum.

(*b*) Lymphatics leading to the lymphatics of the colon and mesentery.

(*c*) Veins leading to the superior mesenteric vein.

(*d*) Subperitoneal connective tissue investment.

(*e*) Peritoneal investment.

(3) Pathological.

Mixed bacterial infection from the intestine.

Abstract Description.[2]

Appendicitis is an infectious exudative inflammation of the appendix vermiformis cæci, originating in any local cause for displacement of the guarding epithelium of the mucosa of the appendix, and progressing by bacterial invasion into the rich layer of adenoid tissue which is under compression.

Bacteria once having penetrated the adenoid tissue remain there and develop for an indefinite period of time and with widely varying degrees of rapidity.

[1] Paper read at the Eleventh International Medical Congress, in Rome, September, 1893.

[2] Photomicrographs by Dr. J. C. Smith and pathological data verified by Dr. H. T. Brooks, at the Pathological Laboratory of the New York Post-Graduate Medical School.

Paper read at the Pan-American Medical Congress, September, 1893.

The natural course of the inflammation is protracted and marked by slow erosion of the mucosa and adenoid tissue, caused by the pressure of exudates and infiltrates.

A more rapid destructive process follows passive choking of the inner tube within the outer tube when exudation into the adenoid tissue is excessive.

The most rapid destructive process occurs when the muscular sheath irritated to the point of spasm contracts firmly down upon the swollen inner tube.

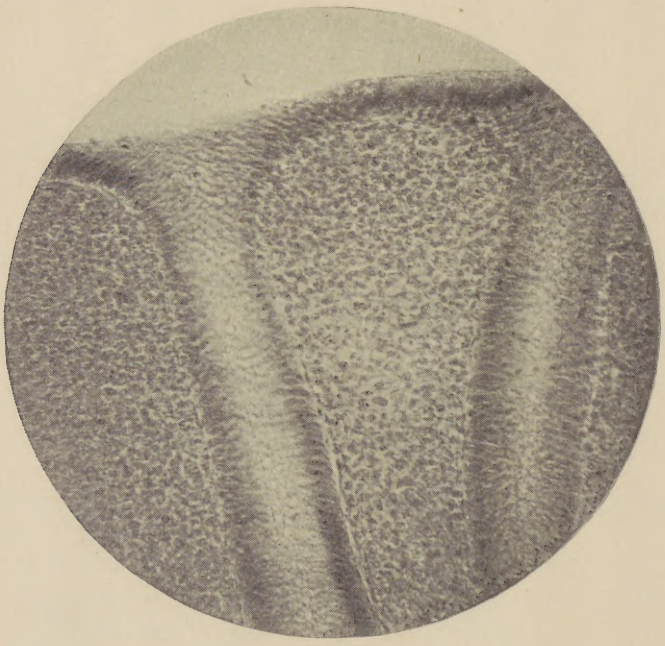

FIG. 1.—Section of normal mucosa and adenoid layer of appendix, showing follicles. × 600.

Incidents in the course of the disease are: (1) Infection along the lymph channels from the appendix to the colon, causing typhlitis, paratyphlitis, perityphlitis and cæcitis. (2) Extension of thrombosis along the veins of the appendix, resulting in mesenteric thrombo-phlebitis, pyle-phlebitis, portal embolism, and abscess of the liver. (3) Local peritonitis from direct infection of the peritoneal investment of the appendix. (4) General

peritonitis from infectious thrombi in the veins of the portal system, and from poisoned peritoneal exudates. (5) Local abscesses in subperitoneal connective tissue.

Detailed Description.

The thin mucosa of the appendix vermiformis cæci is composed of a single layer of columnar epithelium cells. Beneath the epithelial coat is a layer of adenoid tissue so thick that it often constitutes the principal mass of tissue of the whole appendix.

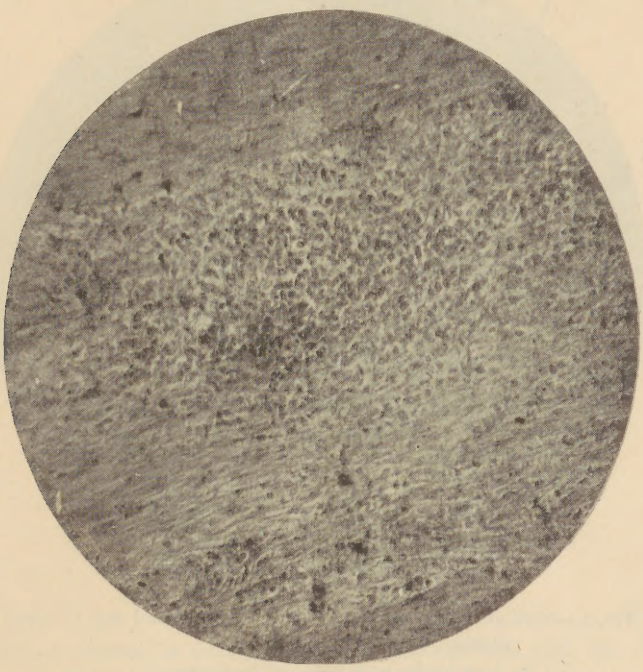

Fig. 2.—Section of muscular coat from mild case of appendicitis, showing infiltration of leucocytes in lymph spaces. × 600.

The layer of epithelium which guards the adenoid layer against infection is easily injured by foreign bodies or fæcal concrements in the lumen of the appendix, especially when violent exercise or a blow causes forcible impact of mucosa against such bodies. I believe that many acute attacks of appendicitis thus follow the efforts of parturition. The epithelial layer of the

appendix is also rendered less resisting when the mucosa of the colon is inflamed from any cause. Thus appendicitis is apt to occur as a complication of typhoid fever or dysentery or cholera, and it is, I think, common as one of the sequelæ of la grippe, although the New York Board of Health reports do not verify

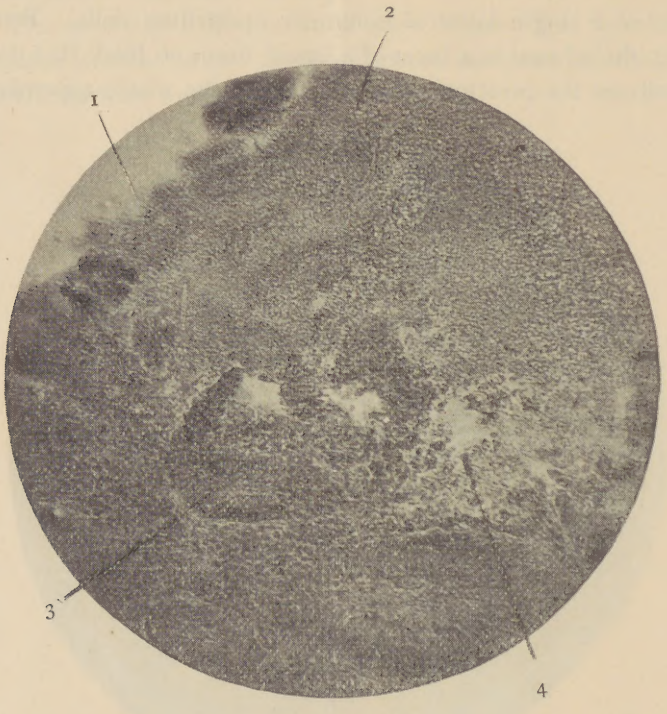

FIG. 3.—Section corresponding to Fig. 1, but from mild case of appendicitis.

1. Free border once occupied by mucosa.
2. Necrotic area.
3. Broken down mucous follicles.
4. Breaking down adenoid tissue. × 600.

this belief. Nematodes and amœbæ coli are probably responsible for some cases of appendicitis, and tubercle bacilli open the gates for other infection. The mucosa and adenoid tissue are easily crushed, as by the efforts of the athlete, compressing his appendix between a full cæcum and the pelvic wall.

The mucosa may disappear in natural retrograde changes of the appendix (Ribbert). When once the epithelial guard has been broken, streptococci, staphylococci and bacilli at once enter the exposed adenoid tissue very much as saprolegnia finds entrance to the tissues of a living fish at a point where the scales have been torn away. All structures of the appendix then become more or less distended with fibrin and serum, while leucocytes crowd the lymph channels.

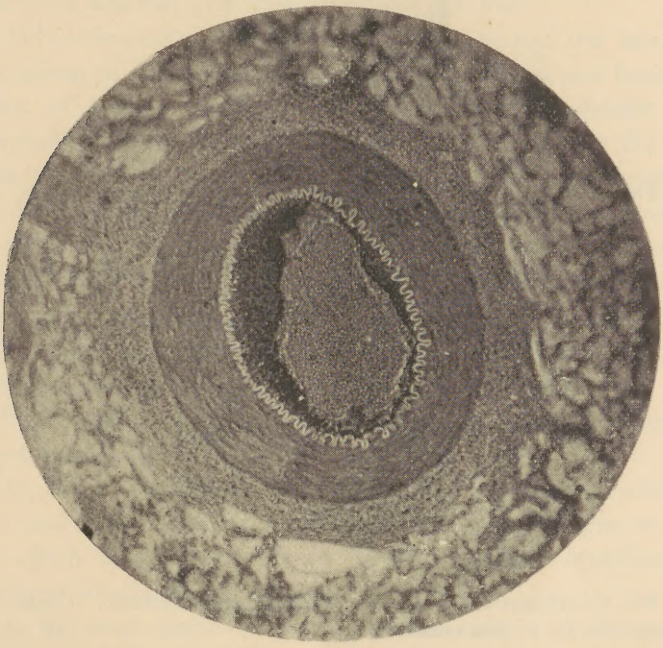

FIG. 4.—Section of solitary artery of the appendix from mild case of appendicitis, showing proliferating endarteritis. × 150.

If the process is a mild one, the mucosa of the appendix is gradually replaced by connective tissue. Necrosis of the adenoid tissue then progresses slowly from obstruction to capillaries and lymph channels, and an erosion of the adenoid tissue eventually bares the inner muscular coat of the appendix. Connective tissue next fills the gaps, the lumen of the tube is obliterated, and the disease is at an end; years, perhaps, having elapsed since its inception.

The appendix is usually supplied by a single artery. If proliferating endarteritis occludes the lumen of the solitary artery the result can be readily foretold.

The appearance of an appendix which is the seat of mild infectious inflammation varies according to the amount of exudates in its tissues. In some far-advanced chronic cases the appendix is hardly firmer in texture or different in appearance

FIG. 5.—Transverse section of a longitudinally split appendix. Exudation and lymphadenoid proliferation moderate.

1. Mucosa and adenoid tissue bulging a little.
2. Submucous connective tissue not thicker than the combined muscular and peritoneal coats.
3. Muscular and peritoneal coats, not hypertrophic.

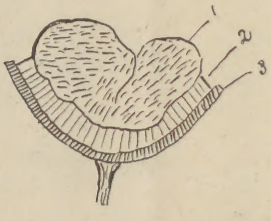

FIG. 6.—Transverse section of a longitudinally split appendix. Exudation excessive.

1. Mucosa and adenoid tissue bulging prominently.
2. Submucous connective tissue about seven times thicker than the combined muscular and peritoneal coats.
3. Combined muscular and peritoneal coats.

from a normal one, and in all probability many a death trap of this sort has been replaced in the abdomen by the surgeon who, in response to the patient's symptoms, has made an exploratory incision and then failed to find what he expected to see. One can no more trust an appendix than he can an egg by external appearance. When exudates distend the tissues greatly the appen-

dix is firm and tense like the erect penis of a child. On making an incision through the outer tube in such a case, the inner tube bulges so prominently into the opening that one can readily understand how great has been the pressure. The inner tube then being split, is usually found to be studded with minute punctate reddish dots and a few ashy necrotic specks upon its free surface, and it has a more pultaceous feel than a normal mucous surface. It is difficult for an untrained eye to discover by gross appearance whether any mucosa remains or not.

When bacteria have finally begun to poison the investing peritonæum of the appendix, exudates are thrown out from the surface of near-by peritonæum to wall in the offender, and absorption of bacterial products may be so rapid that the peritonitis remains quite local, and soon subsides, to reappear at some later period. This is an interesting phenomenon, and rather difficult of explanation unless we assume that increased production of poison is met by increased activity of absorbents of the peritonæum to a point of close compensation, and that the organized lymph, which remains in the form of adhesions, is not so susceptible to later poisoning as the original serosa, each mild attack of peritonitis having then a moderate tendency to protect against recurrence.

In more virulent cases a large mass of peritoneal exudate forms, and this becoming infected breaks down into pus or thin fluid, which is in turn walled in by more exudate furnished by the almost intelligent peritonæum.

In the most vicious cases the peritonæum is so poisoned at the outset that it loses control of itself and cannot build coffer dams.

Bacteria may infect the subperitoneal connective-tissue layers and cause local "cellulitis" with resulting abscess, but this inflammatory process is by no means so common as it was supposed to be in the days when "cellular tissue" was thought to be a good descriptive term.

Thrombi form in the veins of the appendix and its mesentery in the mildest of cases, and these may go on to extensive mesenteric thrombo-phlebitis, with resulting general peritonitis, pyle-phlebitis, portal embolism and liver abscess, in cases in which the appendix has not been held in suspicion at all.

The phenomena of infectious appendicitis vary according to the predominating species of bacteria in any given case, but the pyogenic streptococci and staphylococci do most of the wide infecting.

Violent cases of appendicitis usually attract attention directly to the real source of the trouble when the exudates and infiltrates cause sloughing of small or large portions of the inner tube by

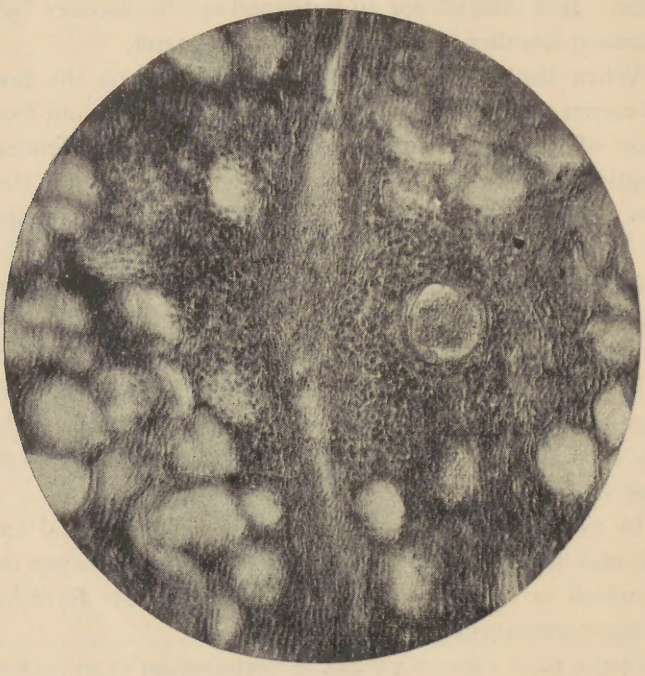

FIG. 7.—Longitudinal section of vein, showing thrombus surrounded by leucocytes, from mesentery of appendix in mild case of appendicitis. × 600.

cutting off blood supply to small parts, or by choking the whole inner tube within the outer tube. At first the pain is reflected to various parts of the abdomen and there is general tenderness of the whole peritonæum, but at the end of twenty-four or forty-eight hours we can, as a rule, find the most marked tenderness at the point made classical by Dr. McBurney in the right inguinal region. We must not forget, however, that the appendix may be down in a scrotal hernial sac, or attached to the liver or to the left ovary. I have no explanation for the fact that the whole

peritonæum is inflamed before the pain becomes localized, excepting on the hypothesis that the peritonæum needs to be pretty thoroughly irritated by poison before it resents the onslaught and walls in the intruder. The cæcum, distended by gas, may hide the tender point in the right inguinal region, and may even mask a large abscess or mass of exudate.

The most acutely painful symptoms occur in those cases in which the muscular coat of the appendix is excited to spasm, for when this happens the intestine contracts in sympathetic spasm at short intervals, causing the most agonizing colic. Fig. 8 represents an appendix which I caught in the act of spasm, and it explains why perforation is more apt to occur at the tip, the exudates in the tissues being crowded to that point, so that the knobbed tip is under great pressure.

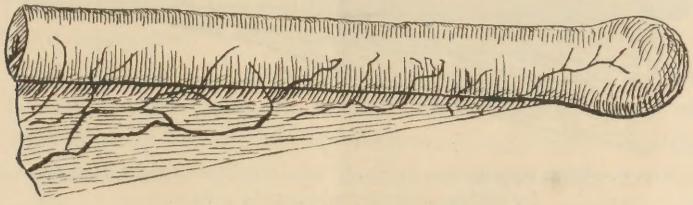

FIG. 8.—Appendix with muscular coat in state of spasm.

Fig. 9 shows the same appendix immediately after the mesentery and muscular base of the appendix with their contained sympathetic nerves had been cut. In this appendix both longitudinal and circular muscular fibres were found to be fully developed clear to the tip, so that the case probably represents an ordinary one.

It is not improbable that the appendix sometimes cuts itself in two by irregular contraction of the muscular coat, just as an annelid or nereid, when held by one extremity, cuts itself away from the enemy. It is not an uncommon experience to come upon a separated appendix or portion of appendix when we are operating, but ordinary circumscribed necrosis may be responsible for this.

Next to the waves of colic, the most distressing pain occurs in the cases in which peritoneal exudates form a solid mass over

the iliac arteries, the blow from the strong pulse there being equivalent to seventy finger pokes per minute against an inflamed abscess wall. If added to this the large nerves of the pelvis respond to irritation with neuralgia, the condition of the patient is deplorable. Infection from the appendix to the walls of the cæcum and colon in mild cases, or in the early stage of severe cases, may cause a constipation from inhibition of peristalsis, or a diarrhœa from the irritation by ptomaines. The infection may be so insidious as to cause perforating necrosis of the colon several inches away from the appendix, and in a case in which the appendix is to outward appearance almost normal. There is apt to be an area of thrombosed veins, however, extending between the

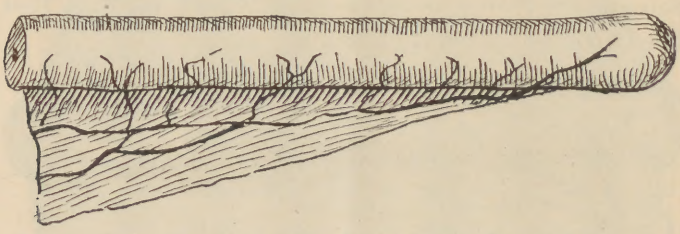

FIG. 9.—Same appendix as in Fig. 8 immediately after relief of spasm by cutting mesentery and muscle at base.

appendix and necrotic bowel. The cases of infection at a distance from the appendix have been classified as typhlitis, paratyphlitis and perityphlitis, according to the particular structure that was supposed to be involved, but as a matter of fact, all structures of the bowel wall are usually involved at one and the same time, and it is not difficult, so far as my experience goes, to trace the infection to its focus at the appendix. I have not as yet, at operation, found any variety of typhlitis that did not evidently begin at this focus. Sometimes infection of the bowel wall takes place by a short route at points where the tip of the appendix touches, but the bowel ordinarily becomes infected by progression of infection along the lymph channels in the loose submucous or subperitoneal connective tissue planes.

Bilious vomiting, while not always present, is a characteristic symptom in all forms of acute appendicitis, and is probably due to a reversing of peristalsis at the duodenum by ptomaines absorbed by the peritonæum and excreted with the bile.

Relapsing cases of appendicitis, more properly called exacerbating cases, are, I believe, of four principal types:

(1) Cases in which the poisoned peritonæum of the appendix responds in various mild exacerbations of local peritonitis. Colic is not a marked feature of these cases, and the septic symptoms are unimportant.

(2) Cases in which the muscular coat of the appendix is excited to the point of spasm from time to time. Colic is the most salient symptom, but septicæmia is neither severe nor persistent, excepting as a result of further complications.

(3) Cases in which small or large portions of the inner tube slough and cause well-marked septicæmia until the sloughs have decomposed enough to escape into the bowel.

(4) Cases in which a chronic abscess cavity fills in exacerbation and empties by slow absorption at irregular intervals. Persistent septicæmia is the chief symptom.

I do not make a classification of perforating cases, because any appendix may perforate at any hour when exudates and infiltrates have caused a sufficient degree of necrosis. Hydrops of the appendix and other odd results of appendicitis are self-explanatory. Exacerbating cases of appendicitis eventually come to an end by slow destruction of the mucosa and adenoid tissue, or by acute inflammatory disaster, or by surgical operation.

I am in favor of early operative treatment in practically all cases of appendicitis, in view of the fact that the inflammation is so infectious in character. So long as the patient chooses to carry about with him a hive of bacteria, he knows not just when or where they will swarm. Moreover, every hour of progress of an acute exacerbation of appendicitis means increased damage to viscera, and the conscientious physician cannot allow that for a patient who trusts him. Many cases of appendicitis run such a mild course for years that the patient is hardly aware of his trouble, and yet the mildest case may end in a disastrous exacerbation at any hour. The simple diagnosis of appendicitis I hold to be sufficient excuse for operation, and as soon as this stand is taken by physicians generally, the patients who would die from the disease under procrastinating treatment will be spared to their

families and friends, to say nothing of saving time and relieving anxiety for patients who have exacerbations. I do not know how any other deduction can be rational.

I have adopted three distinct lines of operative procedure for the requirements of different sorts of cases, and employ three standard incisions.

My first is for cases not complicated by extensive adhesions or pus.

The incision is one inch and a half in length through the right linea semilunaris and all structures of the abdominal wall. It is sufficiently oblique to follow the natural trend of the external oblique aponeurosis. This very strong aponeurosis is, perhaps, the structure most worthy of attention in this vicinity. It must not be pulled away from its slight vascular connections with retractors, or sloughing will occur, and it must be closely sutured to prevent hernia. The incision having been made, the next step in the technique consists in finding a longitudinal muscular ribbon which labels the colon. This ribbon on being found is pulled through the incision until the appendix comes up on the end of it, like an eel at the end of a line, the rest of the colon being instantly replaced in the abdomen as fast as it emerges. The mesentery of the appendix is ligated with small catgut, and the outer tube is snipped through with scissors very close to the cæcum. The inner tube is then ligated with a strand of eye silk well down into the cæcum, and the stump of the inner tube cut short.

The peritonæum round about the stump is scarified until pink serum exudes, and then closed over the stump with three or four Lembert sutures of catgut. If the tiny ligature escapes for any reason it will go into the lumen of the bowel. The plan of simply ligating the stump of the appendix is undesirable for several reasons. In the first place the ligature includes infected tissue, and perforation is quite likely to occur below the point of ligation. When the stump separates it may leave a troublesome mucous fistula if the abdominal wound is treated as an open one, or ordinary signs of perforation occur if the abdominal wound has been closed. If the stump of the appendix heals nicely

under ligature, it leaves a scar which is the weakest point in the intestinal tract; an "Esquimaux window" scar. For these reasons I believe that the ligature will give place to the suture, or, better yet, to the combined suture and ligature as described.

The small wound of the abdominal wall is closed with silkworm-gut sutures which include peritonæum, transversalis fascia, and transversalis and internal oblique aponeurosis, knots being cut short to remain permanently. The external oblique aponeurosis must have its own tier of silkworm-gut sutures. The skin incision is very prettily closed with fine catgut.

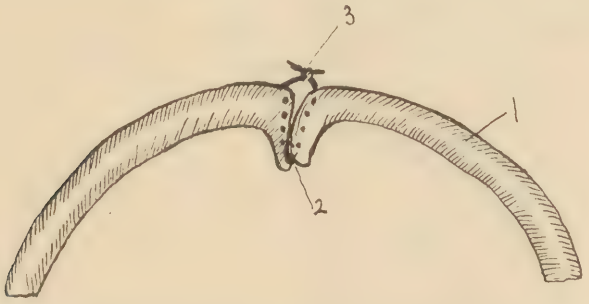

FIG. 10.—Diagrammatic view of stump treated by combined ligature and suture.

1. Transverse section of portion of cæcum.
2. Tiny ligated stump of inner tube of appendix.
3. Lembert suture burying stump. Dotted lines show course of suture beneath peritoneal coat.

My second incision is for cases with extensive adhesions, but without pus or infected exudates. It is twice the length of the first incision, and the three-inch cut extends through the right linea semilunaris as in the first one and in the same direction. The patient is placed in Trendelenburg's position for this operation, so that we can work easily by sight with minimum amount of disturbance of viscera. The wound of the abdominal wall is closed with four tiers of sutures. The first, of silkworm gut for peritonæum, transversalis fascia, and transversalis aponeurosis; the second, for internal oblique aponeurosis; the third, for external oblique aponeurosis, and the fourth, of catgut for the skin. Each layer of aponeurosis must have a separate tier of

sutures, for the reason that its muscle pulls in a separate direction from the others. The silkworm-gut knots are cut short and left permanently.

My third incision, three times the length of the first one, is for cases with abscess or infected exudates, and is made over the point of greatest dulness in the inflamed area, no matter where that may be. The danger of infecting peritonæum is thus lessened, and easy access is gained to the point where most work is to be done. The patient is placed in Trendelenburg's position, and we work by separating adhesions in a direction cephalad from the groin. By this plan of procedure each separate collection of pus escapes outward upon the abdominal wall,

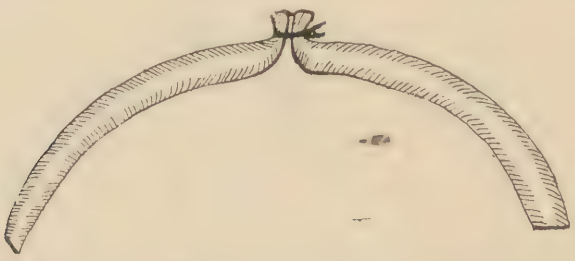

FIG. 11.—Simple but risky method of ligating stump of appendix. Same section as in Fig. 10, but showing ligature of all tissues of unburied stump.

4. Ligature.

while adhesions yet remain cephalad and dorsad to protect the abdominal cavity. As a matter of fact, I have learned by experience to have little fear of pus in the abdominal cavity when it can be reached with saline solution and the drainage wick.

As soon as an abscess cavity is opened it is cleansed with fifteen volume of peroxide of hydrogen, and the septic cavity thus rendered harmless before adhesions are further separated. This is one of the most valuable points in the management of pus cases. The diseased appendix is finally removed and all adhesions loosened. If we rest content with evacuating abscesses simply, the diseased appendix remaining may cause septicæmia or further appendicitis, and adhesions remaining will bind the bowels in faulty position, so that the patient may remain an invalid perhaps for life. We shall have fewer and fewer of these

cases as the advantages of early operation are gradually comprehended, and as the necessity for thorough work in desperate cases is appreciated.

If any pus or infectious material escapes into the abdominal cavity, we must wash it out with warm normal saline solution. (Approximately - ʒizz–Oij.) This will not injure the serosa, whereas common water or solutions of chemical disinfectants are quite sure to injure the serosa and cause vexatious adhesions at least. Common water introduced into the peritoneal cavity has killed many a patient a month or a year later. The peritoneal cavity is the one place in which antiseptics are unnecessary or positively harmful in surgery, but elsewhere throughout the operation an antiseptic technique cannot be too rigid.

In the cases with pus or infected exudates the bowel is usually found to be infected and almost, if not quite, gangrenous in places. Such damaged bowel is sutured with seven-day catgut to the margin of the abdominal wound, so that when sloughing occurs later, or when intestinal contents are to escape, they will appear externally instead of intra-abdominally. The next step is the most important of all in badly-infected cases, and it consists in the introduction of a wick of absorbent gauze as large as one's thumb, surrounded by gutta percha tissue or protective silk, through which small holes have been snipped. The wick is placed with one end down in the pelvic cavity and the other end exposed externally, so that a large absorbent dressing placed over it will suck the peritoneal cavity dry, and keep it sucked dry. The absorbing dressing will sometimes require hourly renewal. An immense amount of offensive peritoneal fluid is discharged through this wick for about forty hours, and as soon as the discharges rapidly decrease the wick is removed. For further drainage we then depend upon osmosis produced by the introduction of deliquescent salts into the bowel. When the wick is removed adhesions will have made a well into which we can pour peroxide of hydrogen until suppuration diminishes, and then the process of granulation can be hurried by the use of balsam Peru.

My statistics in appendicitis cases to date, roughly classified, are as follows:

		Recoveries.	Deaths.
Cases without extensive adhesions, infected exudate masses or pus	28	28	0
Cases of extensive adhesions without infected exudate masses or pus	9	9	0
Cases with infected exudate masses or pus	11	7	4

Two of the deaths were among my first cases, at a time when there was good authority for simply opening their large abscesses and not separating adhesions or removing appendix remains. The abscess cavities were treated antiseptically, but both patients died of septicæmia; one a week after operation, the other about a month after operation. I now know that both patients probably had multiple abscesses. Two of the patients who recovered were treated in the same crude way. One had another attack of appendicitis a year later, and the other is an adhesion invalid in consequence of this bad surgery. In all of the other cases all adhesions were separated and the appendices removed. By far the largest number of the cases were treated within the past twelve months, and among these two deaths have occurred; one from shock in a case with pus and old firm adhesions binding damaged viscera together in a snarl. Resection of intestine should have been done immediately in this case, instead of tedious separation of adhesions. The other death occurred from acute obstruction of the small intestine a week after operation, when I happened to be out of reach. Judging from late experience I feel that all of the forty-eight cases could have recovered under the technique which would now be employed.

Because of the great amount of interest recently shown by surgeons in appendicitis cases, there is a tendency on the part of some to cry "fad," and to quote the extravagance of ovarian surgery. There is, however, a marked difference between following a fad and suddenly awakening to the full appreciation of the fact that through all of the centuries we have been burying unnecessarily a certain class of patients. An inflamed ovary may be a very useful organ. It may yield to treatment, and it seldom threatens life excepting when it is the seat of important

neoma or abscess. An appendix vermiformis, on the other hand is always useless and when infected is a direct and constant menace to life. Its bacteria cannot disappear under medical treatment, though exacerbating symptoms may subside.

Appendicitis has recently been termed the "American disease." I doubt if it is more common here than in other countries, but fear that it may have been overlooked. This assumption is

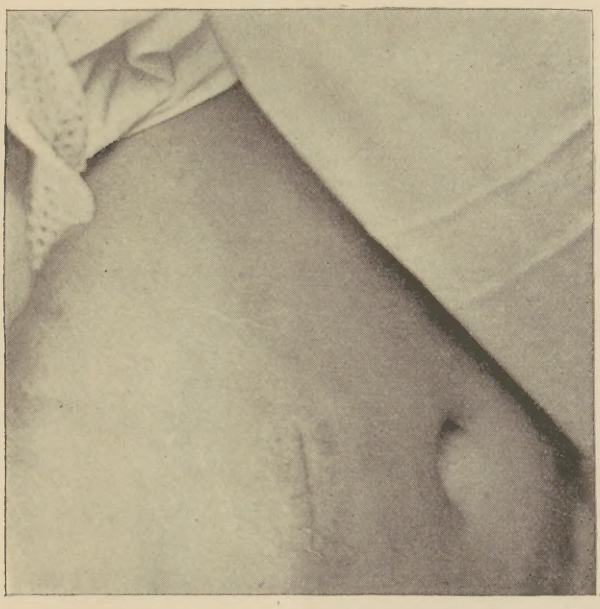

FIG. 12.—Incision one inch and a half in length, ten days after removal of the appendix.

based upon the fact that physicians of my acquaintance who had not made the diagnosis of appendicitis until recently, now find several cases of it in their practice during the year, and are able to call to memory cases which died under a different diagnosis in former years.

The following list of diagnoses had been made in cases in which at operation or at necropsy I found typical appendicitis lesions: Typhoid fever, la grippe, typhlitis, perityphlitis, paratyphlitis, cæcitis, intussusception, idiopathic peritonitis, salpingitis,

ovaritis, gall stones, gravel, bilious colic, bilious fever, bilious peritonitis, malarial fever, psoas abscess, abscess of abdominal wall.

The specimens of appendices which I now present for inspection are not very dissimilar in outward appearance. The first specimen is normal, the second is one in which the mucosa is disappearing. In the third mucosa has gone and adenoid tissue is disappearing. In the fourth neither mucosa nor adenoid tissue remain and connective tissue has taken their place.

Microscopic sections from these specimens, stained to show bacteria and pathological changes wrought by them, are here exhibited.

I have not, as yet, been able to secure a specimen showing simple catarrhal inflammation of the appendix, and believe that the term as ordinarily applied is a misnomer.